Home | Online Catalog | Newsletters | Therapy Index | Psoriasis / Eczema | Free Vitamins | Order | Contact Us

High Homocysteine Levels More Deadly Than High Cholesterol, Studies Reveal

A newly recognized and significant risk factor for coronary artery and other vascular diseases has been established within the past decade. Mildly elevated levels of homocysteine have been identified in 21% of patients with coronary artery disease, in 24% of patients with cerebrovascular disease and in 32% of patients with peripheral vascular disease. Researchers conclude that homocysteine is up to 40 times more predictive than cholesterol in assessing **cardiovascular** disease risk.(1-2)

Homocysteine is formed by the body as a naturally synthesized byproduct of methionine metabolism. Like cholesterol, homocysteine performs a necessary function in the body, after which, if the right cofactors are present, it will eventually convert to cysteine and other beneficial compounds such as ATP, cysteine and S-adenosylmethionine (SAM). When left intact, it enters the bloodstream and begins attacking blood vessel walls, laying the foundation for heart disease, stroke and other **cardiovascular** diseases.(l,2)

Many enzymes, or catalysts are involved in the complete metabolism of homocysteine. If any of these enzymes is defective or functions inefficiently, the body is less able to successfully process homocysteine. This inefficient enzyme function may be due to a mutated or defective gene, identified by Dr. Rima Rozen at McGill University in Montreal.(2) More often this breakdown in metabolism is due to deficiencies of certain nutrients. . .particularly B-6, B-12 and folic acid. (1-3) When this function is disordered, whether due to genetic defect or nutrient deficiency, homocysteine accumulates and enters the bloodstream where it promotes oxidation of lipids, causes platelets to stick together, enhances the binding of Lp(a) to fibrin and promotes free radical damage to the inside of arteries.(l)

Some have suggested that the obvious solution to reducing homocysteine would be to restrict methionine intake by restricting foods such as meats that are rich in methionine. Then the supermarket shelves would be lined with low methionine and methionine-free foods. That makes about as much sense as switching cabins on the Titanic. Methionine is a sulfur-containing amino acid that is involved in the synthesis of protein, important in the maintenance of cartilage, and needed for the formation of other important amino acids such as taurine and carnitine. Methionine is not at fault. The problem is when homocysteine cannot be converted.

Elevated Homocysteine Can Easily Be Normalized

The good news is...elevated homocysteine levels, whether due to nutrient deficiencies or defective genes, can easily be normalized in virtually all cases, simply and inexpensively, using a combination of nutritional supplements. The most effective defense against homocysteine buildup is a combination of vitamins B-6 and B-12, folio acid and trimethylglycine (TMG).(1-3)

There are three biochemical pathways used by the body to reduce homocysteine. In one pathway TMG donates a

methyl group which detoxifies homocysteine. In this reaction, TMG is reduced to DMG (dimethylglycine), that familiar-product sold as a supplement for its energizing effects. In the other routes, folic acid, B12 and B6 convert homocysteine into nontoxic substances.(I)

Some people can't utilize one or another of these pathways. That is why a combination of all these nutrients is most effective for lowering homocysteine.(1,2) In some people vitamin B may not be efficiently converted to its active co-enzyme form, pyridoxyl-5-phosphate. In that case supplementing with pyridoxyl-5-phosphate would be necessary.(3) Isn't it ironic that heart disease and stroke which kill 40% of Americans, may be due to a great extent to malnutrition.

What Is Trimethylglycine?

Trimethylglycine (TMG) is the chemical term for betaine, sometimes sold under the name anhydrous betaine (without water). TMG functions as a **methyl donor** to lower homocysteine and increase beneficial S-adenosyl-methionine (SAM) levels. SAM, a bioactive form of methionine, is a **methyl donor**. It contributes a **methyl** group (CH3) to other molecules to change their activity. A synthesized version of SAM has proven to be valuable for treating a number of conditions including cirrhosis of the liver, depression, osteoarthritis and fibromyalgia.(I)

Methyl groups are thought to protect cellular DNA from mutation. As people age, they often do not have enough available **methyl** groups to safeguard DNA. Abnormal methylation patterns are found in many people with cancer. Eating foods that contain **methyl** groups such as beets, green leafy vegetables and legumes is helpful, but these must be eaten in relatively large quantities several times a week. Therefore, dietary supplements such as TMG may often be necessary to provide the body with sufficient protective **methyl** groups.

Good quality betaine comes from sugar beets through a complex extraction method and should not be confused with betaine HC1 (betaine with hydrochloric acid) which is usually synthesized and commonly used as a digestive aid. There are no published studies on whether betaine HC1 can function as a **methyl donor**. Although it is theoretically possible, its extreme acidity makes it an unlikely candidate for chronic use.(I)

Remarkable Effects of Nutritional Therapy

The remarkable effects of the supernutrient combination (TMG, B6, B12 and folic acid) can be seen in people with genetic defects in enzyme production that cause elevated homocysteine. People with these disorders frequently die of **cardiovascular** disease before reaching adulthood.

In one case history report, a 16-year-old Japanese girl was unable to walk with or without support, and had severe peripheral neuropathy, muscle weakness and convulsions. Her vascular system was on the verge of collapse. B6 or B12 didn't help. Folic acid lowered homocysteine, but didn't improve her symptoms. Two months after adding TMG to the regimen, her homocysteine level dropped and she was able to walk with support. Seventeen months later, she was free from convulsions and able to walk normally again.(1,4)

This case history demonstrates the seesaw relationship between homocysteine and SAM. The girls SAM levels went from undetectable to near normal after the first two months of treatment while her homocysteine levels fell dramatically.

If these nutrients can overcome a genetic disorder, consider how powerful they can be in reducing the risks associated with elevated homocysteine in the general population. Some people who have been taking this homocysteine lowering nutrient combination for more than a decade reported many benefits including fewer colds, more energy, increased endurance and lower blood sugar levels.(I)

Homocysteine Finally Recognized

The homocysteine theory of **cardiovascular** risk was first tested and published by Dr. Kilmer McCully in 1969, but, with everyone focusing on cholesterol at that time, his findings were ignored. Finally, almost 30 years later, the word is out on homocysteine. In addition to *NBC Nightly News with Tom Brokaw,* articles have been published in Newsweek, The Wall Street Journal, The Los Angeles Times, Prevention magazine and more.(I)

Homocysteine levels rise as people age. Therefore, any anti-aging program must take homocysteine level control into consideration. Lowering homocysteine has benefits beyond heart protection. When the blood supply to the heart is blocked, a heart attack results. When blood to the brain is blocked, a stroke results. If the penile artery is occluded, impotence results. Blockages in the extremeties results in intermittent claudication or pain in the affected extremity.(I,2)

Homocysteine and Other Risk Factors

Homocysteines relationship to heart disease may explain some things that cholesterol never could. These B vitamins and homocysteine are so interrelated that homocysteine levels could be used to assess vitamin status. This could explain the increase in heart disease which has occurred in women over the past two decades which coincides with the use of birth control pills. Birth control pills deplete vitamin B6 and raise homocysteine levels. Smoking, a known risk factor for heart disease, also depletes vitamin B6 and smokers generally have low levels of folio acid and vitamin B12...all needed for homocysteine metabolism. Its not surprising that the statistics linking smoking to heart disease are similar to those linking high homocysteine levels to heart disease.(I)

Use of Homocysteine-Lowering Nutrients

For best results, a combination of homocysteine-lowering nutrients should be taken. TMG (anhydrous betaine), folic acid, vitamin B12, vitamin B6 and/or pyridoxal-5-phosphate are the nutrients that have been found to lower homocysteine in clinical studies. They all contribute something along the biochemical pathways which break down homocysteine. While any one of these factors alone may lower homocysteine in some people, the best insurance is to take them all. Based on the latest available research, the basic homocysteine-lowering formula per day is: 500-1000 mg. of TMG, 800 mcg. of folic acid, 500 mcg of vitamin B12 and 50 mg. B6.(1)

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